CLINIC OF DR. HENRY A. CHRISTIAN

PETER BENT BRIGHAM HOSPITAL

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ACUTE VEGETATIVE ENDOCARDITIS

DR. CHRISTIAN to students: I wish you would take the patient in the room under the seats (P. B. B. H. Med., No. 7427) and go over her.

Dr. Christian to another student: I want you to take this patient's pulse (P. B. B. H. Med. No. 7115) first, and when you have finished listen to his heart.

Dr. Christian: The patient here is twenty-eight years of age (P. B. B. H. Med. No. 7115). He was admitted to the hospital on August 20. He has been here continuously since then. He came in complaining of weakness, anorexia, high fever and drenching sweats. He was born in Russia and has lived in the United States for five years. His family history is negative. He has been married for two years. His wife is healthy. He has one child who is healthy. His habits are good. Up to January of this year he worked as a clerk in a small store. The work was not hard nor tiring. Since January of this year he has had a somewhat harder job in a wholesale hardware store. Three years ago he was at the Boston City Hospital with what he thinks was typhoid and pneumonia. He was there for eight weeks. After going home it was four weeks before he could go back to work. He has had frequent sore throats. He has never had rheumatism, scarlet fever, pleurisy or malaria. There has been no known association with tuberculosis. There is no history of any acute infections of any kind other than those I have already given. His past history is negative—he has been a healthy man. He weighed 210 pounds before the present illness began. He has lost weight until his weight was down to 148 pounds just before he came

in. This was a gradual loss of weight over a period of six months. During all of that time he was only a little bit sick. During the last two months before coming in his loss of weight was a little more rapid.

During that time he was more definitely ill; that is, he was having fever, sweats, etc. For three years he has had a cough yielding a small amount of white, clear sputum. He has never been sick on account of this cough. He was never examined by a physician. Six months before he came in (that was about January 1, when he changed his job) he began to have more cough and raised a large amount of thin, watery sputum. He did not go to see a physician at that time, or at least if he did he did not have a physical examination. About June 1, of this year, he rather suddenly developed a high fever in the afternoon which continued and was frequently as high as 104°. At that time he began to get more definitely weak and to have night sweats. His temperature, which was anywhere from 102° to 104° in the afternoon, was more nearly normal in the morning, apparently some mornings it was normal. He was seen then by a physician and was sent to the Jewish Hospital to have a thorough examination. From there he was sent to the Boston Dispensary for an X-ray of his chest. According to him the picture showed definite evidence of pulmonary tuberculosis. He was sent from the Jewish Hospital to Rutland (a State tuberculosis sanatorium). He went home, however, and remained at home for three weeks before he went to Rutland, possibly having some difficulty in getting in. During the three weeks he was at home his temperature was taken regularly and he always has this very high evening fever. At Rutland they were unable to find any pulmonary lesion, and after remaining there for some time he was advised to come back to Boston and go to some hospital for a general examination. Apparently they did not feel that he had pulmonary tuberculosis. He followed their advice as far as going home was concerned and also followed their advice in regard to the hospital and came in here and was admitted on August 20, 1917, and has been here since. Since he has been here he has continued to do apparently what he began to do on June 1; namely, run a fever which sometimes is a continuous fever without much fluctuation and at other times is a more definitely fluctuating temperature. There has been very little change in that

since he has been here. (Demonstrations of charts.) You see sometimes these up and down excursions are very great and at other times the temperature is very uniformly up. That is shown better in the four-hour chart (Fig. 1), a sheet of which I will pass around. The temperature at times has been as high as 105° and 105.5° in the afternoon. In the morning it is frequently normal; it is occasionally subnormal. So there is a fever of known long duration. Apparently it was pretty accurately taken before he came to the hospital from June 1 to August 20, and there has been a continuation of the same from August 20 to date, November 5.

Dr. Christian to students: What do you make out in regard to the pulse and to his heart?

Student: The pulse is normal but the artery does not come up well.

Dr. Christian: It is a small pulse but there is nothing abnormal about the pulse wave. It is a soft pulse and probably a little rapid, but not a particularly rapid pulse. How about his heart?

Student: The heart sounds are a little muffled. I did not hear any murmurs.

Dr. Christian: The heart sounds are a little muffled, but there are no murmurs to be heard. How about the size?

Student: It goes away out into the axilla.

Dr. Christian: By percussion or palpation?

Student: By palpation.

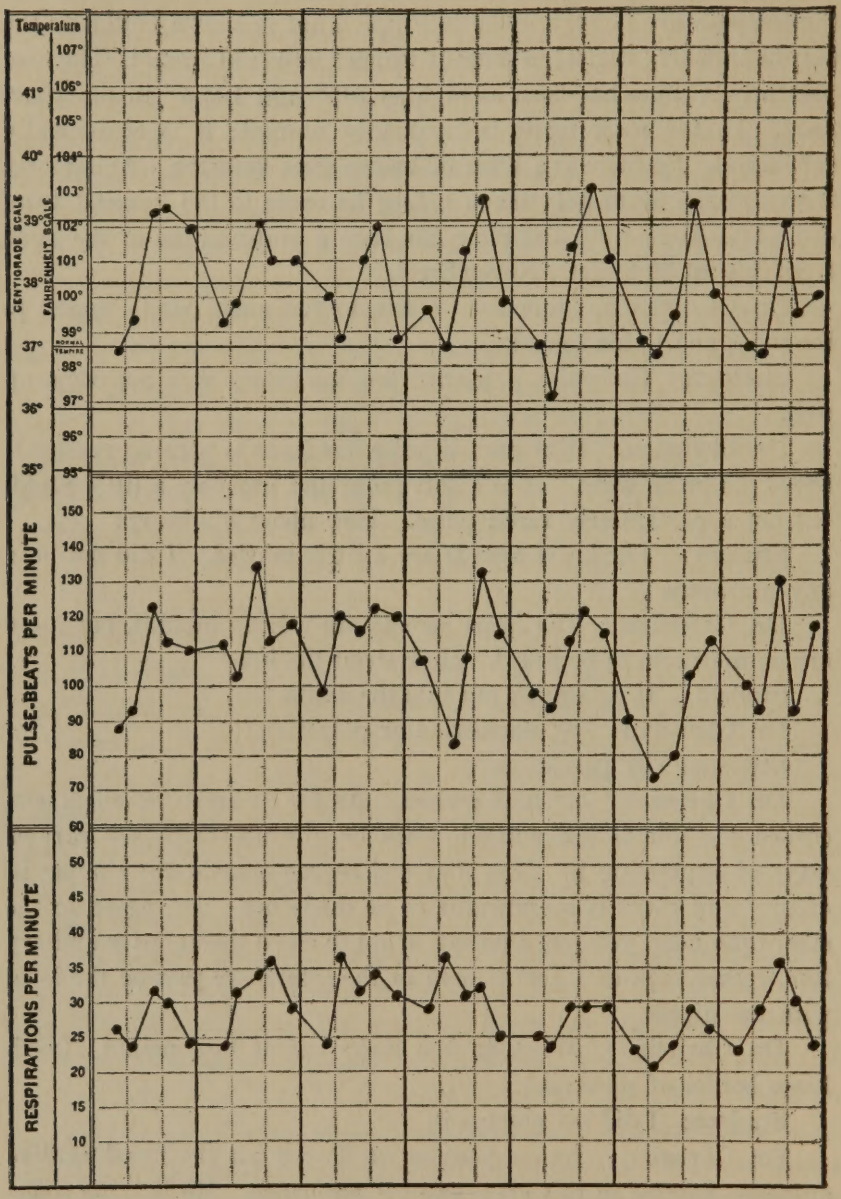
Dr. Christian: That is correct. As far as auscultation is concerned the sounds are muffled, weak or distant—there are various ways of expressing it. The first and second sounds are similar in quality and there is no murmur to be made out. In regard to the size of the heart you feel a pulsation out to about the anterior axillary line, rather high as I feel it here in the interspace just on the level with the nipple.

Dr. Christian: What do you make out at that point? (Left lower border of sternum.)

Student: I did not percuss it.

Dr. Christian: As we percuss we do not get the usual cardiac dulness—flatness in this area—we get resonance. He is lying somewhat tipped on the left side and he generally lies over on that side.

Fig. 1.



Four-hour chart of patient P. B. B. H. Med. No. 7115.

Dr. Christian to patient: Can you lie flat in bed?

Dr. Christian: He has a bad knee and that interferes with his motions. It may be that his heart is an easily movable one and when he is lying over on his side it tips over and that is suggested because the impulse is high, whereas ordinarily with an enlarged heart you find the impulse lower down. To put it another way, a normal-sized heart as it goes over is rotated somewhat on a fixed point here at the base, and although normal sized the apex comes up as it is tipped over.

Dr. Christian to student: See if you can locate the impulse.

Student: It is always well to take into consideration the position of the patient, whether lying on one side or the other, in locating the apex or the border on percussion because the heart in a perfectly normal individual moves a good deal. In percussing with the patient first on one side and then on the other side you find the apex normally shifts about that (three to four cm.) far.

Student: I cannot locate the apex impulse very satisfactorily.

Dr. Christian: It is pretty hard to tell where it is. It is a weak diffuse one. What about the one out here—is that gone?

Student: Yes, the impulse felt when the patient was not flat in bed has disappeared.

Dr. Christian: Now listen and see where you get the sounds best. Sometimes when it is difficult to locate the heart by percussion as to borders or by palpation you can check up your impressions by listening and getting the point at which the sounds are heard best, more distinctly or louder, remembering that sometimes consolidated lungs or fluids transmit sounds so that you hear heart sounds very distinctly outside the cardiac area, so that in an area of dulness you cannot localize always the position of the heart by the sounds.

Student: About here.

Dr. Christian: It is about in the normal position when you listen to the sounds.

Student: There is a low murmur.

Dr. Christian: Did you say you heard a murmur?

Student: I thought I heard one on expiration, but I do not know.

Dr. Christian: As we percuss that heart with the patient flat on his back the border of dulness is, quite definitely, just in the nipple

line. In other words, really his heart was shifted over somewhat but was not enlarged essentially. It got into normal position with him flat on his back. There is still resonance instead of absolute dulness over the precordial area but the dulness is greater than it was before, so the heart probably is not enlarged or if enlarged it is only very slightly enlarged. You can hear a soft, slightly blowing, systolic murmur just over the apex, but it is very slight. I won't bother you to go over his lungs. I will speak of the few findings we have had there. There is a slight occasional râle and a little dulness at the bases.

The very considerable loss of weight is evidenced in his forearm. You remember he said he weighed 210 pounds last January, and that is not the arm of a man that weighs 210 pounds by a good deal now. When he came in his physical signs were practically what they are to-day. He showed loss of weight and he showed a heart that was normal in size. The day he came in a soft, systolic, blowing murmur was heard over the precordium. His blood-pressure was $\frac{150}{85}$. He had a hæmoglobin of 74 per cent., red cell count of 4,256,000 and a leucocytosis of 14,400. Those have been his physical signs in very large part ever since he came in. On the next day, August 21, examination of the heart was negative; there were no murmurs; abdomen was negative; spleen was not felt; liver edge was easily felt on inspiration. On the 23d the note was made, "Pulmonary signs negative except for occasional fine crackles throughout the right chest front and back. These râles, however, are not constant. Sounds of the heart are rapid and of good quality. Systolic murmur heard over the precordium, of greatest intensity at the base. Liver edge felt from the fourth interspace to second fingers below the costal margin in the right nipple line. Spleen not palpable." On August 29 this note was made, "Sounds rapid, regular, tic-tac quality. No murmurs. Percussion of both lungs shows slight relative dulness over the lower portion, front and back, and on deep respiration a few fine crackles are heard. Respiration on the whole shallow and not rapid. Patient not cyanotic. No areas of bronchial breathing. Respiration in general rather faint and distant." The notes read, as far as physical examination is concerned, just like those made by various members of the staff, who have gone over him since he came in on August 20.

Two X-rays of his chest are negative. One was taken on August

21, and the other one was taken on September 14. We had a third plate, taken quite recently, which showed that the heart is not enlarged. The diaphragm is rather high and possibly the heart border is a trifle out to the left, but there is no very definite enlargement, and there is no change in the heart size in the different plates. There is the normal amount of thickening along the bronchial tree, but the apical portion of both lungs and the peripheral portion of both lungs are entirely clear, and there is no evidence of infiltration of any kind. The third plate was not one in which the patient was lying squarely on his back. It showed a definite deformity due to the fact that the sternum had rotated to one side and the vertebræ to the other side, and near the sternal margin there was a round area which was pretty dense and a little bit larger than the end of my thumb, and the rest of the plate was entirely negative. The question comes as to whether that round area of increased density was more than what we ordinarily see in plates because in this region there is the regular density of the hilum of the lung and frequently enlarged peribronchial lymph nodes, and it may be that that particular point in the rotation was brought out in the view. I am a little inclined to think that that interpretation should be made and that all three plates bringing it down to a pretty recent date are negative.

As to special examinations—an ophthalmoscopic examination made several times is negative. During his stay in the hospital he has been running a varying leucocyte count: 14,400 when he came in, one count which was as high as 38,700 on October 7, and two or three days later than that, on October 10, a count of 10,800. Those represent his extreme variations. Blood cultures were taken on August 21, September 8, September 13, and on October 19, and all of those blood cultures were negative. His sputum has been repeatedly examined recently. In early tests there was very little sputum. These examinations have been negative for acid fast organisms. There is nothing particular about these sputum examinations. Recently he has begun to cough up bloody sputum. This is some sputum that he coughed up this morning. You see it is mostly blood, but in it are grayish masses of pus. That specimen has not been examined yet for tubercle bacilli. That development of bloody sputum is a quite recent affair. (Subsequent examination showed no tubercle bacilli.)

The only other thing that has happened during his stay in the hospital is the development of some fluid in his abdomen in the latter part of August, and in the first part of September. The fluid accumulated enough so that he had shifting dulness, typical signs of fluid, and on September 13 we tapped his abdomen and got out 2750 Cc. of straw-colored fluid, with a cell count of 2400; 99 per cent. lymphocytes and 1 per cent. polymorphonuclears. Smears made from the clot of that specimen showed no tubercle bacilli, and a guinea pig injected was negative for tubercle bacilli. Just prior to that he began to develop some signs suggesting a meningeal irritation, a positive Kernig sign, some headache, etc., so that on September 10 lumbar puncture was done and a normal amount of fluid was obtained, having a normal cell count and a slightly positive globulin test. Since his abdomen was tapped the fluid has not reaccumulated. Subsequently he has had no more signs of meningeal irritation. The Wassermann reaction in blood and spinal fluid is negative. The Von Pirquet skin reaction is negative.

You notice that he has a bandage on his forearm just above the elbow and I spoke of his having some trouble in his knee. Those represent subcutaneous abscesses which have been drained by incision, and the one on his knee, which was opened about October 25 or 26, showed an organism which grows as very small colonies. They grow in the bouillon as a flocculent precipitate, and if you shake the tube you see little masses of bacteria that float. Culturally and morphologically the findings justify the diagnosis of streptococcus viridans. Those organisms were obtained from the deep abscesses in the region of his knee, remembering that his blood cultures have been negative.

The only other change that has taken place in him is that the liver has quite definitely enlarged. It was made out on the first examination in the hospital as enlarged, the edge being felt not very much below the costal margin. Since then it has increased in size and this morning was noted to be tender. It is not a very large liver but it is one that extends pretty well down into his abdomen and recently has been tender. The spleen has never, I think, been felt. The other day in feeling his abdomen I felt something to the left of the median line which I thought might have been spleen but followed across, it seemed more like an enlarged left lobe of the liver than the

spleen, so it is fair to say that as far as physical examination goes the spleen has never been felt.

The patient's essential feature has been the fever. The fever has existed since June 1 to date and it has been an up and down fever. His physical signs have been essentially negative—there is no definite evidence of a cardiac lesion, no definite evidence of a pulmonary lesion, occasionally a few râles are heard in the lung, but they have been inconstant. After the patient has been lying flat on his back there is some slight dulness at the base of the lung, and râles there. X-ray examination showed no pulmonary lesion.

The question comes up as to what is the probable diagnosis in a patient with that type of temperature and so few physical signs. It practically always lies between three things: syphilis, miliary tuberculosis and vegetative endocarditis. Syphilis is the least frequent cause of that type of temperature and there is nothing in this patient, either Wassermann reaction or history, to suggest syphilis. Probably miliary tuberculosis is the more common cause of that type of temperature. In this patient we have a negative guinea pig test on his abdominal fluid as far as tuberculosis is concerned, a negative Von Pirquet reaction, which when negative in the presence of fever is not worth very much, and it happens to be negative here, and a spinal fluid which was normal, showing no evidence at that time of tuberculosis of the meninges, and an ophthalmoscopic examination which showed no miliary tubercles.

A short time ago it was suggested that it might be of interest to have a complement fixation test done for the reaction with tubercle bacilli as antigen and we sent his serum up to Saranac Lake, where they are doing a good deal of work on that reaction, and got back the report that they would be very much interested to know what would turn out eventually in the case, but the serum was negative for tuberculosis. The other thing is that miliary tuberculosis usually, if it is producing the temperature, produces very definite physical signs in the lungs or signs of meningeal tuberculosis within a shorter period of time than the interval between June 1, when his symptoms began, and November 5 (to-day), and none of those signs have appeared. When he got the stiff neck, etc., we thought he was beginning to have meningitis, but no progress has been made on that score.

It is of interest to go back and see what diagnoses were suggested as the patient went along. On August 20 a house officer, after taking the history and making a physical examination, thought it was miliary tuberculosis. On August 21 one visiting physician said: "My diagnosis would be diffuse tuberculosis, probably pulmonary in origin and miliary now." On August 29, when I saw him, my interpretation was: "The temperature curve and its duration even without any cardiac signs except enlargement strongly suggests acute endocarditis as the most probable diagnosis." There are some other notes here in which no definite diagnosis is made. Another house officer said on September 18: "No diagnosis possible at this time." A little later this same house officer thought: "Recent fluid in the abdomen and general doughy feel of the abdomen rather suggests tuberculous peritonitis." On October 23 he said: "All signs taken together seem to point to a generalized tuberculous infection." On the 24th after some râles were found he added: "These findings seem to strengthen the diagnosis made on the patient of tuberculosis." November 1 I made a note: "The case has continued with fluctuating fever, no development of physical or pulmonary signs by physical examination or X-ray plate. The diagnosis of vegetative endocarditis seems most probable and is in accord with the findings of streptococcus viridans in a deep abscess about the knee joint."

The next patient (P. B. B. H. Med No. 7427) is a young woman, age twenty-four, who comes in complaining of heart trouble. Her family history is negative. Her habits are good. Her work has been light. She has been a milliner and has had rather long hours and not very good conditions surrounding the work, but for six months she has not been at that work because the one who employed her went out of business. She was born in Massachusetts and has lived here always. She had measles at six; tonsillitis at nine; chicken pox at eleven; mumps at eighteen. She has had mild attacks of pleurisy in the left upper chest, the attacks occasionally lasting two or three days, during recent years. She has had no other acute infectious diseases. Her tonsils were removed at the age of nine. She cut her left hand on a piece of glass at the age of fourteen, requiring suture, and the wounded finger has been stiff since then. A year ago she weighed 104 pounds; two weeks ago she weighed eighty-four pounds. This loss of weight has

been gradual. She was quite well up to two months ago, when she gradually began to complain of weakness. She has slept poorly at night. She has felt so weak that she has had to lie down most of the day. She knows of nothing that was the cause of this weakness. That is, the onset was gradual and insidious and she cannot attach any definite event or cause to the beginning. She had not overworked and had not caught cold. After this weakness began she noticed a considerable amount of palpitation of the heart at night and with the palpitation she often had night sweats. These have been getting less marked recently. One month ago she went with a girl friend to the Boston Dispensary and was told at that time that she ought to go to a hospital and was told to go home and go to bed. She did so and was in bed for about two weeks. She did not feel much better as a result of that, so she came to the hospital and was admitted on October 19, 1917. She has had a fever since she has been in the hospital, in some observations it being a little above 102° in the afternoon and in the morning a little above 99°, that is, since she has been here her temperature has not been at any time normal, but it has not been elevated more than about 3° above the normal. You see from the four-hour chart (Fig. 2), like the other patient, it has been a fluctuating temperature, but there have been no such steep rises and falls as in the preceding case.

Dr. Christian to student: What did you make out?

Student: The pulse is regular and very rapid. It is full and easily palpable. The arteries are normal. There is a distinct venous pulse in the neck. The heart is not enlarged to the right, being about 3cm. from the mid-line to the right; to the left I could not percuss it—dulness goes clear over to the side of the chest. At the apex there is a pre-systolic thrill.

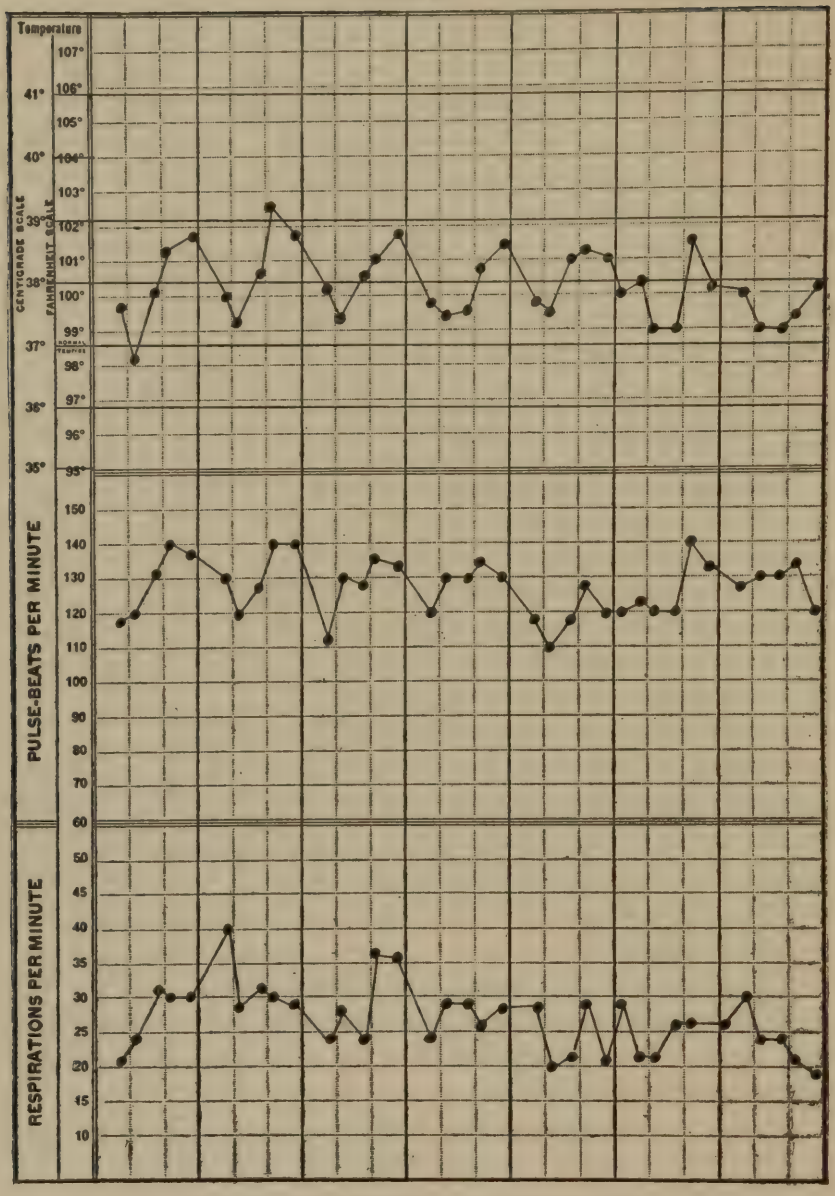
Dr. Christian: There is a systolic murmur heard over the whole precordium, maximum at the apex, and a pre-systolic murmur heard at the apex. There are some râles in the left chest. What did you hear at the left sternal border?

Student: Nothing more than I said.

Dr. Christian: Listen right over the lower left border. What do you get there?

Student: I hear a systolic murmur.

Fig. 2.



Four-hour chart of patient P. B. B. H. Med. No. 7427.

Dr. Christian: Is it systolic? No, there are two murmurs—the loudest and longest is diastolic. The shorter one is systolic. The patient has quite marked murmurs. There are murmurs at the apex, murmurs at the base, and a thrill. There is a systolic murmur that you can hear both at the base and at the apex, and from the middle of the sternum at about the level of the third interspace down along the left sternal border there is a loud, blowing diastolic murmur and a quite evident, shorter systolic murmur of about the same intensity, so that the patient has quite definite signs of a valvular lesion producing changes. In addition she has a definite anæmia of the secondary type; a hæmoglobin of 65 per cent. and a red count of 3,400,000 when she came in on October 20, and on the last day of October the hæmoglobin had dropped from 65 per cent. to 51 per cent. and the red count had dropped from 3,400,000 to 2,128,000. In addition she has a polymorphonuclear increase, giving a leucocytosis the highest 17,400 and the lowest 9200; another count 14,200.

When she came in her heart action was made out to be regular, rapid, heart enlarged, loud systolic and diastolic murmurs heard over the whole precordium; in the third interspace one inch to the left of the sternum loud and musical in quality. These murmurs are heard in the aortic area and along the left border of the sternum, with less intensity at the apex. On October 20 I made a note: "In the aortic area and over the sternum and along the left sternal border to the tricuspid area is heard a double murmur which in places becomes high pitched and musical in quality. The same murmur can be heard with less intensity at the apex. The first sound has a ringing valvular quality." On October 25 one of the visiting physicians made out the murmurs as follows: "At the apex can be heard a definite pre-systolic roll which is transmitted somewhat toward the sternum. Just inside the apex can be heard a short, soft, systolic murmur and a similar diastolic murmur. The diastolic murmur becomes much more prominent toward the base and is very loud in the third left interspace close to the sternum. The systolic murmur can be heard in this region, but is considerably fainter than the diastolic. The pulse is Corrigan in type. No capillary pulsation can be made out."

That patient has a weakly positive Wassermann reaction in her blood. A blood culture was taken on October 26. The plate showed

about twenty-five colonies—small colonies—and here are transplants from one of those colonies and on the blood serum surface you will notice very minute dry colonies, that appear just as little points, slightly glistening and scattered over the surface. You notice in the bottom of the bouillon tube a sediment which if you shake it gives some flocculent masses of precipitate, rather fine masses in suspension. That organism is characteristic of streptococcus viridans and as far as we know is streptococcus viridans.

These two cases present certain features in common and certain distinguishing characteristics. In the first place they both had an insidious, gradual onset, as far as we can make out from the histories of the patients, not marked by any definite cause and not a sudden onset. Both patients have lost weight. Both patients have had night sweats. Both patients have had fever of an irregular type, high in the afternoon. There is nothing very definite in the past history of either individual to point to a beginning of an infection. One has been surprisingly well. The other patient, the woman, has had scattered along during her childhood and young girlhood the ordinary children's diseases. She had tonsillitis at nine, and her tonsils were taken out at nine. One patient shows essentially a normal heart and the other physical signs are normal, with the exception of an enlarged liver and an occasional râle in the lung. The other patient shows marked signs of a valvular lesion of the heart, pointing to aortic insufficiency and mitral insufficiency and possibly a mitral stenosis. During the last few days the physical signs of mitral stenosis have become a little bit more evident. The important thing in that case is the diastolic murmur, pointing to either aortic insufficiency or mitral stenosis or both, and that is what a diastolic murmur means in a patient and is always important. A systolic murmur in most cases means regurgitation from the mitral valve and is ordinarily in itself of very little importance. Both patients show leucocytosis, one shows very little anæmia and the other one shows striking anæmia which has increased since the patient has been in the hospital. The first patient has had repeated negative blood cultures, but streptococcus viridans was isolated from pus in a deep subcutaneous abscess in the region of the knee. The other patient, the young woman, has a positive blood culture of streptococcus viridans. Recently the man has added to his

picture increased sputum with a considerable amount of blood, making a true hæmoptysis. The condition in each is probably primarily due to the same thing; namely, vegetative endocarditis, the vegetations being due to streptococcus viridans. In the one case the vegetations apparently have been very minute and have caused no ulcerative or destructive process in the valves and up to the present have not interfered with the function of the muscle. In the other case, because this patient apparently prior to the onset of this illness had nothing pointing to a valvular lesion in the heart (I say "apparently" in the second case), the organisms have caused destruction of the valve, as far as the aortic valve is concerned, with aortic insufficiency; possibly similarly a destructive process of the mitral valve, with mitral insufficiency; possibly some reparative process going on in the mitral valve; possibly large masses of vegetations, such as we have seen recently in an autopsy, have interfered with the function of the mitral valve in the sense of producing an obstruction and giving us for one reason or the other slight signs of mitral stenosis.

The woman has had no peripheral evidences of bacterial infection, nor has she had any signs of emboli from the valves getting out into the peripheral circulation except a few petechiæ in the conjunctivæ. She has had no petechiæ on the skin, no abscesses, no embolic circulatory symptoms, etc. The man for a long time presented the same picture and had no signs of emboli getting away from his heart until the infection developed first around his elbow and later around his knee, which can be explained as bacterial in origin, possibly just a small amount of bacteria getting out into the peripheral circulation, lodging at that point and producing a suppuration.

In regard to the hæmoptysis, he has had no extensive cyanosis, no dyspnœa, no extensive pulmonary symptoms or signs, and it is possible that the hæmoptysis has taken place owing to the organism from his heart getting into his lungs and producing some sort of destructive process. There is still the possibility that he has a tuberculous lesion and that the tuberculous lesion is causing the usual ulcerative type of process and that he has hæmoptysis from that cause. It is not very probable that that is the kind of hæmoptysis that we have spoken of in cases of mitral stenosis or chronic myocarditis, examples of which have been shown to you, where the hæmoptysis is due to chronic passive

congestion or infarction, a process that takes place when an embolus lodges in the lung in chronic passive congestion. This may be infarction in the sense of an infected embolus causing interference with the circulation to a part of the lung with some breaking down, but it is not the ordinary form of infarction with chronic passive congestion.

However, I think both cases are of the same general nature and the man particularly well illustrates the difficulty in making a diagnosis in one of these cases and the long time that the process can continue without producing any demonstrable cardiac lesion, though the primary cause is acute endocarditis. That has been pointed out by various observers and the first thing to-morrow afternoon I will run over briefly some of the points on the general subject of these long-continued cases of vegetative endocarditis. They are important and a very interesting group, particularly as to mistaken diagnoses, and a very common happening is for them to be called tuberculosis and the patient be sent to a tuberculosis hospital, as occurred in this man.

November 6, Dr. Christian: The first thing this afternoon I want to finish up a little bit more in detail the discussion of the cases that I showed you yesterday. That type of case that I showed has been variously named. Sometimes it has been spoken of as a case of "chronic infectious endocarditis." That was the term under which Dr. Billings reported a group of them in the *Archives of Internal Medicine,* in 1909, vol. iv, page 409; and, by the way, that is a very good discussion of the subject to read. There is a great deal of interest in it. The discussion itself is very brief, taking only four or five pages, but there are fourteen cases of the condition reported with not extensive notes but pretty full notes on the essential features of the condition. Then other terms have been used, such as "sub-acute bacterial endocarditis." That is the term that Dr. Libman, in New York, has used. There are two papers, one in the 1912 and the other in the 1913 volume of the *Transactions of the Association of American Physicians,* by Dr. Libman on the same subject, with a considerable number of case reports. Dr. Libman is particularly interested in the group during the bacterial free stage and Dr. Billings speaks more of them when bacteria are found in the circulation. Then in the *Quarterly Journal of Medicine,* 1909, vol. ii, page 299, Sir William Osler gives a very good description of the group. Then you will find

in these various papers a discussion of the organism that causes the condition—the organism which I spoke of as being streptococcus viridans. You will find that Billings in his paper speaks of the organisms as a type of pneumococcus. The organisms were studied at that time by Dr. E. C. Rosenow and he was inclined to regard them as a pneumococcus organism. Libman in his various papers does not give it any special name or discussion as to just what it is. They have been described occasionally as the coccus of rheumatism. There has been a great deal of discussion as to the exact classification of this organism. They are cocci that grow in chains, and present certain cultural characteristics which we ordinarily regard now as indicating one of several varieties of streptococcus. So do not feel disturbed in reading different papers to find different names given to the organism. Another term that is used very often for the diseased condition is "chronic ulcerative" or "chronic infectious endocarditis." This term really describes the condition and its varieties. Sometimes they are chronic; sometimes they are fairly acute. Sometimes there is a destructive process in the valves with signs of valve lesions, such as shown by one of our cases, and there are cases in which the vegetations are small and there are no destructive lesions, as illustrated by the other case.

A very interesting thing about the cases is the mistakes in diagnosis—the different views in regard to a diagnosis before the correct diagnosis is made. That was illustrated to a certain extent by our own case, which was sometimes regarded as a case of tuberculosis—miliary tuberculosis—and at other times as a case of endocarditis. Not infrequently the patients are regarded as cases of typhoid fever as another possibility. Not infrequently, on account of the fluctuating temperature, they are thought to be malaria. Those various diagnoses are made.

Another striking thing about the cases is that very often the condition exists for a long time with the patient in really a pretty good condition; that is, notwithstanding the fact that there is bacteræmia, fever and an active valve process going on, compensation is perfectly good, the patient feels pretty well. For instance one of Dr. Billings' cases during the febrile period went on a vacation and did some duck shooting and fishing, and many of the cases are able to travel, some

going off on long trips, etc., so that the cases may exist with relatively few symptoms. Most of the cases are eventually fatal. Even those cases that become bacteria-free and apparently remain free from bacteria for several years eventually die. I think of Dr. Libman's cases, in this second report, of the twenty-one cases which he had observed, seventeen had died during the period of observation and the diagnosis was confirmed in sixteen of those seventeen by autopsy findings.

The condition is somewhat different from what is ordinarily spoken of as malignant endocarditis, or malignant vegetative endocarditis, where the process is much more rapid, the destruction of the valves is much more extensive and the patients live a shorter period of time. The second case which I showed you is more of that type.

If you take more frequent blood cultures I suppose it is much more certain that you would get positive results but fairly frequent blood cultures may give negative results in these cases. Most of the patients have a leucocytosis, but a good many of them have a normal white cell count, so the presence or absence of a leucocytosis is not characteristic of the condition; sometimes it is present, sometimes it is absent. The most difficult cases to diagnose are those that present no cardiac symptoms and no cardiac signs.

Now, of course, the patients that I have shown you as illustrations of that may turn out to have something else and that not be a correct diagnosis of this type of vegetative endocarditis. If that is the result I will let you know later, but I have seen cases with just as little physical signs and just as few symptoms referable to the heart who eventually died, and autopsy showed vegetative endocarditis with small vegetation on the valves, and the patients did not die for a very considerable period of time after they began to run a temperature, and it was only in the end stages of the condition that they began to have septic emboli thrown off and a septic condition develop in various parts of the body, by which the condition was recognized as a vegetative type of endocarditis; and in that connection are particularly interesting the cases that Dr. Libman reports from the Mt. Sinai Hospital, in New York, where the patients did not die of cardiac disease. Some of them died a nephritic death. There is a type of glomerular lesion associated with this particular organism in this particular group of cases. The

organism in this group apparently produces a type of glomerular lesion, one described by Aschoff, in Germany, and since by Baehr, who described it while abroad, but he is now associated with Dr. Libman at the Mt. Sinai Hospital. Some of those cases present themselves with the picture of an enlarged spleen and secondary anæmia. They are called cases of splenic anæmia and the true condition is missed. Some of those cases progress downward along the lines of increasing anæmia and die the death of an anæmic, and then, of course, you get others who have chronic valvular lesions associated with or developing as the result of the vegetative process and they die the ordinary cardiac insufficiency type of death.

November 28, Dr. Christian: The first patient (P. B. B. H. Med. No. 7115) showed in an X-ray taken on November 8, an area of wedge-shaped consolidation with the apex of the wedge toward the hilum. He continued to cough up bloody sputum. Repeated examination of this failed to show any tubercle bacilli but numerous streptococci were present. The liver continued to enlarge and it seemed as if the diaphragm was pushed up quite high on the right. Exploratory puncture, however, in this region yielded no pus. The patient continued to grow progressively weaker and died on November 22 without any additional symptoms developing. Permission for autopsy was refused.

The second patient (P. B. B. H. Med. No. 7427) progressively lost ground without developing any other signs or symptoms of embolism or infarction except blood in the urine, and died on November 14. Autopsy showed acute, large, friable vegetations on the aortic and mitral valves with destruction of part of the aortic cusps. There was a slight old thickening of the mitral flaps with thickening and shortening of the chordæ tendineæ. Infarcts were found in the spleen and kidneys.